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THE GEOGRAPHIC DISTRIBUTION OF DISEASE

III. A DECADE OF POLIOMYELITIS IN LOUISIANA[1]

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The finding of a definite concentration of cases of St. Louis encephalitis in the St. Louis area in weedy places about sewage-polluted streams (*1*) prompted the study of the geographic distribution of a somewhat similar disease, poliomyelitis, in Louisiana. The files of the State Department of Health for the decade ended January 1, 1939, contain reports of 676 cases, practically all of which, it may be assumed, are actual instances of paralysis, since it is not the custom in Louisiana to report abortive cases.

Detailed maps of the 64 parishes in Louisiana were obtained from the Departments of Agriculture and Home Economics of Louisiana State University. Parish health unit directors and their staffs and other interested physicians generously took the time to secure and plot the exact home address of each patient at the time of the onset of the disease, thus circumventing the custom of rural residents of giving the nearest town as their home address. The population of all incorporated communities, wards, and parishes in Louisiana was obtained from the United States Census of 1930. The population of unincorporated communities was obtained from a commercial atlas and was corrected, whenever this was possible, by data in the possession of the State Department of Health.

Accurate data were thus obtained for 59 parishes, in which the distribution of the population and of the cases of poliomyelitis,

[1] From the Department of Pathology and Bacteriology of the School of Medicine of Louisiana State University, and the Louisiana State Department of Health.

according to incorporated and unincorporated communities of various populations, was as follows:

TABLE 1

Size of community	Number of places	Total population	Poliomyelitis cases		x^2/m (3)	Rate per 100,000 population
			Actual	Expected		
	Incorporated places					
Under 100	1	27	0			
100–499	50	17,603	12	6.0	*6.00*	68.2
500–999	42	30,467	23	10.3	*15.65*	75.5
1,000–1,499	25	29,651	24	10.0	*19.60*	80.9
1,500–1,999	15	25,888	24	8.7	*26.90*	92.7
2,000–2,999	14	27,681	21	9.4	*14.31*	75.9
3,000–4,999	18	68,629	29	23.2	1.45	42.2
5,000–49,999	12	117,822	36	39.9	.38	30.6
50,000–499,999	2	535,417	128			
	Unincorporated places					
Under 100 (rural)		890,208	286	301.4	0.79	32.13
100–499	195	[1] 43,342	[1] 26	[1] 14.7	[1] *8.68*	[1] 60.0
500–999	37	[1] 22,561	[1] 7	[1] 7.6	[1] .05	[1] 31.0
1,000–1,999	9	[1] 16,448	[1] 2			
Total		1,825,374	618	618.0	93.81+	33.9

[1] Based on estimated populations and estimated community boundaries.

Statistically significant preponderances of poliomyelitis were found in both incorporated and unincorporated communities of 100–499 population. The highest preponderances were found in incorporated communities of 500–2,999 inhabitants, the maximum incidence being reached in communities of 1,500–1,999 inhabitants (table 1). The increase in incidence from rural communities to communities of 1,500–1,999 population, and the decrease from communities of this size to those of 5,000–49,999 population, were orderly and formed a unimodal curve when plotted (fig. 1).

It was of interest to find that rural areas, when interpreted as unincorporated communities of less than 100 inhabitants, and urban communities of 5,000–49,999 inhabitants had the same low incidence of poliomyelitis. The incidence in the only two large cities in Louisiana corresponded with the incidence of the disease in smaller cities, but the limited number of cities does not permit adequate analysis. Unincorporated communities of over 500 inhabitants, which for the most part are industrial in Louisiana, had an incidence of poliomyelitis similar to that of cities and rural areas and significantly lower than the incidence for incorporated communities of the same size. The possible explanations for this discrepancy will be considered later in this paper.

The age distribution of the cases of poliomyelitis in this series does not differ from the age distribution in other reports, the mode being 2 to 3 years and the mean age 7 years. Variations in age, however, do not explain the preponderance of the disease in small incorporated

communities. The average age of the patients in rural communities of less than 100 inhabitants was 6.0 years; of the patients in New Orleans, where the disease was endemic, 6.8 years; of the patients in Shreveport, where the disease was largely epidemic, 6.9 years; and of the patients in small towns of 500–2,999 inhabitants, 7.2 years. All of these age distributions had the same modal points, and the mean ages showed no statistically significant differences. Because of the lack of age variations, immunological differences probably do not account for the preponderance of poliomyelitis in small towns.

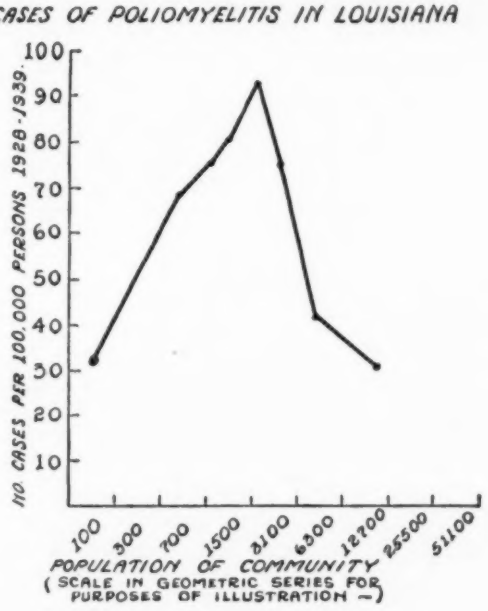

FIGURE 1.

The variations in incidence cannot be explained on the basis of either sex or race. Three hundred and sixty-four patients were male and 324 female, which is a statistically insignificant sex difference and compatible with the usual preponderance of males among children. The racial distribution in small towns is shown in the following table, there being no significant difference between the three types of communities in this regard (Chi square=3.44, $n=2$, $P=0.21$):

TABLE 2

Size of community	Actual values			Expected values			d^2/m (3)
	Colored	White	Total	Colored	White	Total	
Under 100	99	178	277	89.6	187.4	277	1.46
500–2,999	23	64	87	28.1	58.9	87	1.37
3,000–499,999	41	99	140	45.3	94.7	140	.61
Total	163	341	504	163.0	341.0	504	3.44

In summary, analysis of the incidence of poliomyelitis in Louisiana over a decade revealed the same low incidence of the disease in urban and rural (under 100 inhabitants) communities. There was, however, a significant preponderance of cases in incorporated communities of 500–2,999 inhabitants. This preponderance formed a unimodal curve with a peak at the communities of 1,500–1,999 and could not be explained on the basis of age, sex, or race.

The only characteristic in which small incorporated towns differ from rural communities and which might have a bearing on the matter seemed to be the presence of a water supply and the absence of a sewage disposal system. Unincorporated towns and rural communities dispose of human excreta by slow desiccation in the open air,

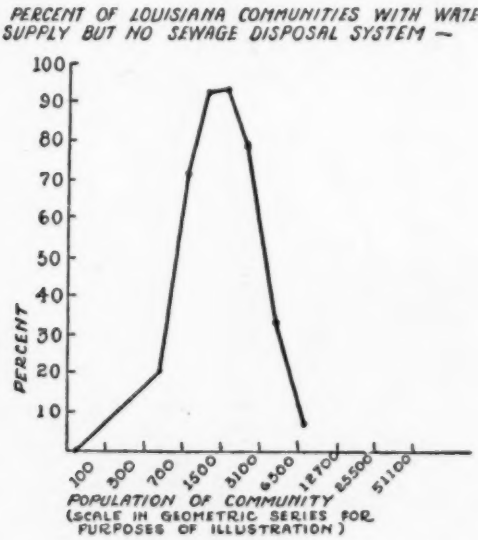

FIGURE 2.

and the first attempt in modernization is usually the construction of a water supply system which disposes of human excreta in a liquefied form and permits the emulsions to accumulate and stagnate in ditches.

A study of communities in Louisiana in regard to water supply and sewage disposal [2] was undertaken from official data obtained through the courtesy of the Bureau of Sanitary Engineering of the State Department of Health. The percentage of communities of various populations which had water supplies but no facilities for sewage disposal was plotted, and the result was a unimodal curve which had a skew in the ascending limb, a modal point, and a sharp descending limb. This curve coincided remarkably with the curve for the preponderance of poliomyelitis (fig. 2), and the correlation was significant.

[2] This study had been under way for more than a year and was in preparation for publication when the recent interesting article by Paul, Trask, and Culotta (8) appeared.

A further analysis of the data revealed that in those communities in which the daily per capita supply of water was less than 10 gallons, the rate of poliomyelitis for the decade was 32.1 cases per 100,000 inhabitants. In those communities in which the daily per capita supply was 10–49 gallons, the rate was 64.1 per 100,000 inhabitants; in communities where the daily per capita supply was 50–89 gallons, the rate was 120.0; and in communities in which the daily per capita supply was 90–500 gallons, the rate was 39.0 per 100,000 inhabitants, which is approximately the rural-urban rate. This observation, which is statistically significant, was true only of communities without sewage disposal.

The rate for poliomyelitis for the decade was over 120 per 100,000 (maximum rate, fig. 3) in 16 communities in Louisiana with more than

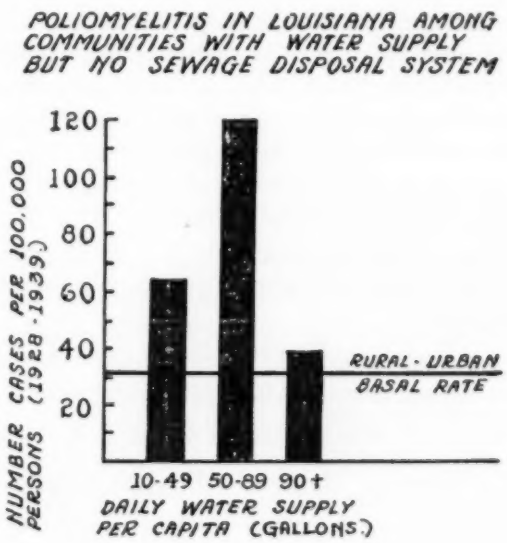

FIGURE 3.

500 inhabitants. In 8 of these communities, in which the population was 1,500–2,999, the average per capita daily water supply was 64.6 gallons. In the other 8, in which the population was 500–1,499, the average per capita daily water supply was 42.0 gallons.

In contrast to these communities, there were 37 communities with populations of more than 1,200 inhabitants in which the rate for poliomyelitis for the decade was less than the rural-urban rate of 32 per 100,000 inhabitants. Nineteen of the communities with populations of 1,200–2,999 reported no cases, and in these localities the daily per capita water supply averaged 170.1 gallons. The other 18 communities, in which the population was 3,000 or more inhabitants and the rate for poliomyelitis was less than 32 per 100,000 (averaging 17.8), had an average daily per capita water supply of 101.4 gallons. The

low rate for paralysis in unincorporated communities with more than 500 inhabitants, which were usually industrial, was associated with an average per capita water supply of over 200 gallons daily. Inasmuch as the daily per capita water supply was for the most part independent of the size of the community, the data suggested the effect of large amounts of fluid as a dilution factor or a factor increasing the rate of flow (fig. 3).

SUMMARY

Poliomyelitis seemed to be widely distributed in Louisiana during the decade under investigation, but the preponderant incidence was in small towns. The question therefore arises as to whether the epidemicity of the disease during the past 50 years has not been influenced by the growing tendency of communities to liquefy excreta without making adequate provision for the disposal of the accumulated fluids.

The 64 incorporated places in Louisiana without community water supply and sewage disposal had 12 cases of poliomyelitis, or 39.7 cases per 100,000, which approximates the basal rural-urban rate. The 27 incorporated places in Louisiana with both water supply and sewerage system had a population of 691,881, and 184 reported cases of paralysis, a rate of 26.6 per 100,000 inhabitants. The 87 unincorporated places in Louisiana with water supply but no sewerage system had 120,811 inhabitants and 101 cases of paralysis, a rate of 83.6 per 100,000 inhabitants.

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NATURAL INFECTION OF *TRIATOMA HEIDEMANNI* WITH *TRYPANOSOMA* CRUZI IN TEXAS [1]

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INTRODUCTION

Previously the writer has demonstrated the natural infection of *Triatoma gerstakeri* with *Trypanosoma cruzi* in the State of Texas (*10*). The object of the present communication is to show that *Triatoma heidemanni* ("bloodsucker," "Mexican bedbug," "kissing bug") is also naturally infected with *Trypanosoma cruzi* in Texas, and, therefore, that this blood sucking insect represents another potential vector for spreading Chagas' disease in man and in animals.

[1] The writer is indebted to Dr. Charles Philips and his colleagues at the Scott and White Clinics, Temple, Tex., for their aid during the collection of live *Triatoma heidemanni*.

Field studies.—In October 1937 and September 1938, the writer was sent by the Public Health Service to Temple, Tex., to investigate the epidemiological significance of *Triatoma heidemanni*. A single adult *Triatoma* previously received from there had been proved to be naturally infected with *Trypanosoma cruzi* (see fig. 1).

Over 150 insects collected at Temple, Tex., were identified by Mr. H. G. Barber, of the United States Department of Agriculture, as *Triatoma heidemanni* (*2, 5, 11*). These bugs were collected chiefly in homes in different sections of the city. The nymphs [2] were usually found in mattresses and bedding and occasionally on the wallpaper or between cracks of wood in bedrooms. Most homes in which the insects were located were fairly modern and the people living therein were of modest means. A few adults and two lots of nymphs were collected in fields.

Laboratory studies.—The technique used in this study has been described (*10*) and consists of (1) demonstration of natural infection of *Triatoma* with trypanosomes; (2) use of experimental animals and methods of inoculation; (3) microscopic examination of the blood for demonstration of infection of test animals; (4) staining of trypanosomes.

EXPERIMENTAL DATA

Natural infection of Triatoma heidemanni with trypanosomes.—About 65 percent of 150 *Triatoma heidemanni* collected during 1937 and 1938 in Temple, Tex., were found to be naturally infected with trypanosomes. All adults examined were infected. Five lots of nymphs representing 42 *Triatoma* collected in different homes were free from flagellates, while of 2 lots of *Triatoma* representing 6 adults and 44 nymphs collected in a cotton field near a farmhouse all were infected with trypanosomes.

Two adult *Triatoma heidemanni* found in Three Rivers, Tex., were likewise infected with trypanosomes. These two bugs were found among about 200 specimens of *Triatoma gerstakeri* collected during 1938 in the same locality.

The insects naturally infected with trypanosomes harbored the flagellates in their intestines and eliminated some of them in their fecal excretion. The flagellates were not found in the saliva of 10 adult *Triatoma heidemanni* examined. The parasite had the morphology of crithidia with long flagellum, herpetomonas, and slender metacyclic trypanosomes; all these forms were often found in the fecal material of both nymphs and adults. The forms of the trypanosomes observed in cover-glass and stained preparations were identical with

[2] The young *Triatoma*, which are known as nymphs, have no wings and cannot fly. They stay near or around a location where a supply of blood is available. The nymphal stage lasts several months. The nymphs then mature, acquire wings, and are able to fly. "Flying tick" stage lasts 1 or 2 months. The adult female lays eggs which hatch within 3 weeks. The adults are usually found in Texas during the months of May, June, July, and August. They are rarely found later.

similar preparations derived from naturally and experimentally infected *Triatoma megista* and *Triatoma gerstakeri* (*10*) and were infective to guinea pigs, mice, rats, and rhesus monkeys (see table 1).

TABLE 1.—*Cultural and microscopic findings in the animals inoculated with the intestinal contents of Triatoma heidemanni*

Key No.	Experimental animal	Source of inoculum	Microscopical examination for trypanosomes in blood		Cultural attempts		Autopsy findings		
			Number of days after inoculation	Results	Number of days after inoculation	Results	Number of days after inoculation	Degree of lymphocytic infiltration (myocarditis)	Leishmania-like *Tr. cruzi* in tissues
335-1a	*Mus musculus*	*Triatoma heidemanni* (Fecal material).	23	+	23	+	23	0	+
335-1b	do	do	13, 132, 184	+++	184	+	184	++	-
335-2	do	do	6, 160, 184	000	184	+	184	+	-
352-1a	do	*Mus musculus* 335-1a	108	+	108	+	108	++	-
352-1b	do	do	108	0	108	+	108	+	-
392-1a	do	Rhesus monkey 388-1	3, 11	00	11	+	11	+	-
392-1b	do	do	11, 202, 308	000	308	+	308	+++	-
399-2a	do	do	60, 139	00	139	+	139	+	-
1413-1a	do	*Triatoma heidemanni*	53	0	53	+	53	0	-
1413-1b	do	do	53, 68, 89	0+0	89	+	89	+	-
1419-4a	do	do	58, 63	00	63	+	63	0	-
1419-4b	do	do	58, 83	00	83	+	83	++	-
1455-4c	do	do	11, 24, 109	000	109	+	109	+++	-
1456-1b	do	do	3, 11, 24	000	24	+	24	0	-
1456-1c	do	do	11, 24, 109	000	109	+	109	+++	-
1461-1a	do	Rhesus monkey 1442-1	52, 282	+, 0	282	0	282	+++	+
1461-1b	do	do	52, 282	+, 0	282	0	282	+++	+
1504-2a	do	*Triatoma heidemanni*	59, 63, 100	0, +, 0	100	+	100	++++	+
1504-2b	do	do	59, 63, 169	0, 0, 0	169	0	169	++	-
1504-2c	do	do	59, 63, 169	0, 0, 0	169	+	169	+	-
1504-2d	do	do	59, 63, 169	0, 0, 0	169	?	169	+++	+
1505-1a	do	do	48	0	48	+	48	+	-
1505-1b	do	do	48	0	48	+	48	+++++	+
1505-1c	do	do	48	+	48	+	48	+++++	-
1578-3a	do	do	30, 63	0, +	63	?	63	+	+
1578-4a	do	do	30, 63	0, +	63	?	63	++++	+
1578-4b	do	do	30, 63	0, 0	63	+	63	+++++	-
1599-3	do	*P. eremicus* 1579-1					[1] 22	++	+
1579-1a	*P. eremicus*	*Triatoma heidemanni*	30	+	30	+	30	+++++	-
1579-1c	do	do	30, 37, 63	+, +, 0	63	+	63	++	-
1598-1a	do	*P. eremicus* 1579-1	33	0	33	?	63	+	+
1599-2a	*P. leucopus noveboracensis.*	do	33	0	33	+	33	++	-
1599-2c	do	do	33	0	33	+	33	0	-
1599-1a	*P. polionotus polionotus.*	do	33	0	33	+	33	++	+
1600-1a	*Rattus norvegicus.*	do	7, 33	0, 0	33	+	33	++++	-
1600-1b	do	do	7, 33	0, 0	33	+	33	+	-
1600-1c	do	do	7, 33	0, 0	33	+	33	++	+
334-2	Guinea pig	*Triatoma heidemanni*	58	+	58	+	58	0	-
334-3	do	do	132, 275	0, 0	132, 275	++	275	0	-
1572-2	do	do	12, 170	0, 0	12, 170	+	170	++	-
1600-2a	do	*P. eremicus* 1579	127	0	127	+	127	++	-
1600-2b	do	do	127	0	127	+	127		
388-1	Rhesus monkey	Culture from 334-2	12, 35, 165	+, +, 0	12, 35, 165	+++	165	++++	-
1442-1	do	Rhesus monkey 388-1	47, 60	+, +	47, 60	++	60	++++	-
1460-1	do	Rhesus monkey 1442-1	133	0	133	+	133	++	-
1597-3	do	*P. eremicus* 1579-1	127	0	127	+	127		

[1] Dead.

Animal inoculations and demonstration of Trypanosoma cruzi in the blood of experimentally infected animals.—Sixty-eight susceptible animals were inoculated with the fecal material derived from seven

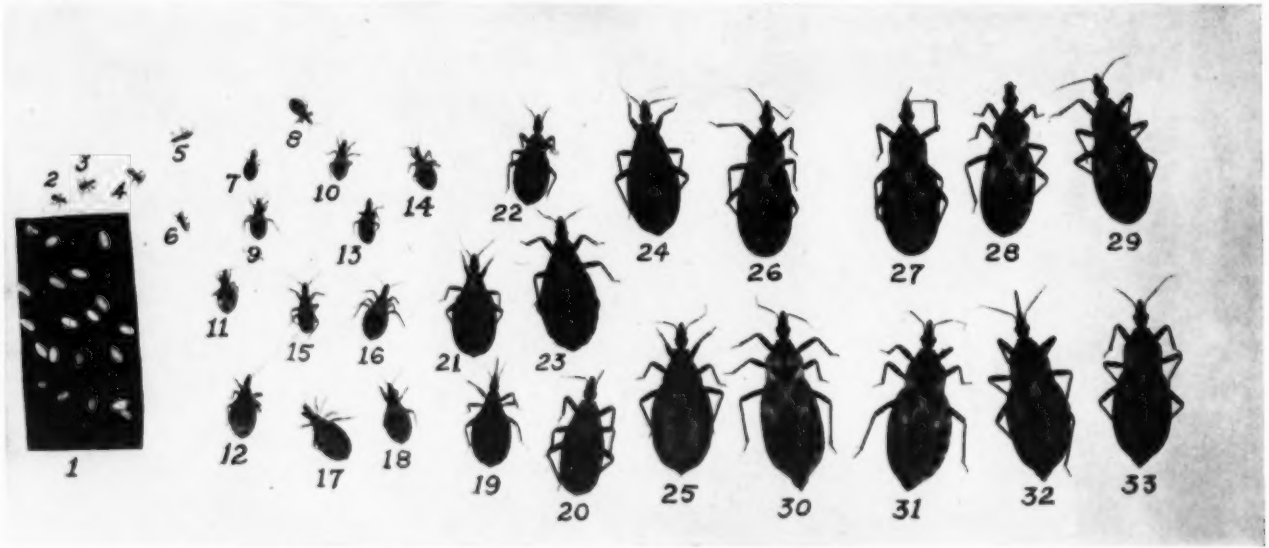

FIGURE 1.—*Triatoma heidemanni* (photographed natural size). 1. Eggs. 2 to 6. Youngny mphs 10 days old. 7 to 10. Young nymphs after feeding on animals. 11 to 18. Nymphs after second feeding. 19 to 25. Large nymphs engorged with blood. 26 to 29. Adult males. 30 to 33. Adult females.

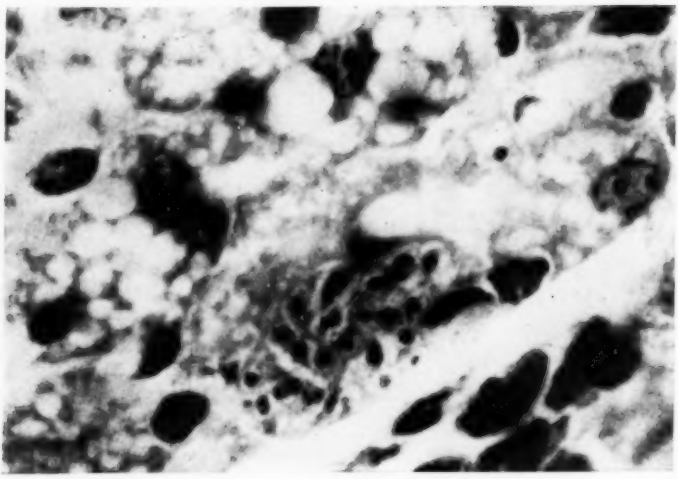

FIGURE 2.—(Photomicrograph) Intracellular form of segmenting *Trypanosoma cruzi* in fat cells from *Mus musculus* 335–1a (table 1). Note segmenting leishmania-like trypanosomes with round or ovoid pale basophilic macronuclei and densely basophilic rod-shaped blepharoplasts. Lillie's modification of Romanowsky's stain. (× 1600.)

groups of naturally infected *Triatoma heidemanni* or with the strains of trypanosomes isolated from these bugs. For the sake of brevity only 46 animal inoculations will be described in this communication, of which 34 are mice (28 *Mus musculus*, 3 *Peromyscus eremicus eremicus*, 2 *P. leucopus noveboracensis*, and 1 *P. polionotus polionotus*), 3 rats (*Rattus norvegicus*), 5 guinea pigs, and 4 monkeys (*Macacus rhesus*). Occasionally the blood of the inoculated animals was examined microscopically under cover glass (objectives Nos. 21 and 45, ocular 10X). Trypanosomes were seen in the blood of 15 out of 45 animals (table 1). The number of trypanosomes seen in any given preparation rarely exceeded 3 per microscopic field (45 x 10). Often over 5 minutes search was necessary in order to demonstrate a single trypanosome in a cover-glass preparation. Once about 5 trypanosomes were found in the blood of a mouse which had been inoculated with 0.3 cc. of a rich culture of *Trypanosoma cruzi*. The movements and the morphology of the trypanosomes in the peripheral blood of test animals were similar to those previously observed (*10*), which are characteristic of *Trypanosoma cruzi*.

Culturing trypanosomes in vitro.—Growth of trypanosomes resulted in 42 out of 49 cultural attempts; of the remaining 7 negative cultures, 4 were contaminated with bacteria. Trypanosomes were found by microscopic examination in the blood of only 8 animals (total of 10 tests) at the time blood was taken. No trypanosomes were demonstrated during 5 minutes of microscopic search in the blood of the remaining 31 animals (total of 32 tests) at the time of cultural attempts, yet rich cultures of trypanosomes (*in vitro*) were obtained from all of these animals. (See table 1.)

The cultural forms, crithidia, herpetomonas, metacyclic trypanosomes, dividing forms, and rosettes were similar in size, morphology, and movements to the forms found in a previous study (*10*). Monthly or bimonthly subcultures were made *in vitro* from each strain for several generations. Some of these strains have been kept *in vitro* on Novy and MacNeal's media for over a year (14 generations). They grow luxuriantly and form colonies on blood agar slants and produce typical infection in susceptible test animals. The cultural forms stain readily.

Gross and microscopic pathology.[3]—Among 64 inoculated animals, 1 mouse died after 22 days of illness (1599–3). The remaining 63 animals were sacrificed at various intervals; the minimum duration of infection was 11 days and the maximum 404 days. At autopsy no pronounced macroscopic changes were noted. The heart blood from each animal was introduced into N. N. tubes for cultural studies and

[3] The writer is indebted to Dr. Ralph D. Lillie and Dr. L. L. Ashburn, Division of Pathology, for their cooperation in this work and reports of histopathological findings.

pieces of tissue and organs were fixed in 10 percent formalin, or occasionally in saturated solution of mercury bichloride containing 10 percent formalin or in 20 parts of formalin and 80 parts of 95 percent alcohol. These were sent to the Division of Pathology. After dehydration of tissues they were imbedded in paraffin and sections stained by Lillie's modification of Romanowsky's stain. All the slides were examined by either Dr. Ralph D. Lillie or Dr. L. L. Ashburn, and by the writer.

Leishmania-like segmenting trypanosomes were found in only 11 cases out of 64 autopsies. These forms were found usually in the cardiac muscle fibers in the atrium (7 cases), in skeletal muscles (3 cases), and in scattered fat cells (1 case). (See fig. 2.) The number of segmenting forms of *Trypanosoma cruzi* in a given cell varies from very few to many. These forms contain a round basophilic macronucleus and densely basophilic rod-shaped blepharoplasts. Lymphocytic infiltration, mostly in the atrium, was noted in 51 cases. Marked myocarditis was noted in 34 animals.

DISCUSSION

The flagellates found in *Triatoma heidemanni* produced infection in experimental animals similar to the infection produced by the strains of *Trypanosoma cruzi* isolated directly from human sources. The morphology and movements of the flagellates both *in vitro* and *in vivo* are also indistinguishable from known strains of *Trypanosoma cruzi* (*8, 10*). From the experimental data on hand it is concluded that flagellates found in *Triatoma heidemanni* are *Tr. cruzi*. (See table 1.)

The present study shows also that cultural tests are most valuable in diagnosing *Trypanosoma cruzi* infections in experimental animals (*6, 7, 8, 10*). Cultures give positive results even when one is unable to demonstrate a single trypanosome in peripheral blood after a long microscopic search. Positive cultures were obtained from about 85 percent of inoculated animals. (See table 1.) A few cultures which have been recorded as negative were contaminated with bacteria and molds. If Novy and MacNeal's media are prepared properly and care is taken to prevent evaporation of water of condensation from test tubes, nearly 100 percent positive cultures may be obtained.

Only 10 of 64 animals showed leishmania-like forms of trypanosomes in muscle fibers, but this finding is sufficient to conclude that trypanosomes isolated from naturally infected *Triatoma heidemanni* are capable of producing this condition in experimental animals. It was interesting to note that in one case leishmania-like forms of *Trypanosoma cruzi* were also found in the fat cells. (See fig. 2.)

There are about 15 species of *Triatoma* known to exist in the United States. Of these, 4 species, including *Triatoma heidemanni*, have been found naturally infected with *Trypanosoma cruzi* (*2, 3, 5, 10, 11*).

Triatoma heidemanni is already a "domesticated pest" in certain homes and causes discomfort to the inhabitants. These insects, collected in homes and in bedding, were all free from *Trypanosoma cruzi* infection, suggesting that the individuals from whom the bugs obtained blood were not infected. The *Trypanosoma cruzi* infection is not transmitted through the egg, and newly hatched nymphs are free from the infection. Such nymphs remain free from infection if they feed on normal individuals. *Triatoma* collected outside homes in Temple were found, however, to be infected with *Trypanosoma cruzi*. These bugs often get in homes during the months of May, June, and July, and thus represent a potential source of infection.

SUMMARY

1. The reduviid bug, *Triatoma heidemanni*, popularly known as "blood sucker," "Mexican bed bug," and "kissing bug," collected in or around dwellings in Temple, Tex., was found to be naturally infected with *Trypanosoma cruzi*. This blood-sucking insect has already become a household pest in certain localities and represents a potential vector for spreading Chagas' disease.

2. The strain of *Trypanosoma cruzi* collected in Temple produced infection in monkeys (*Macacus rhesus*), mice (*Mus musculus*), American deer mice (*Peromyscus eremicus eremicus, P. leucopus noveboracensis, P. polionotus polionotus*), rats (*Rattus norvegicus*), and guinea pigs.

3. Cultural tests proved to be very fruitful. Out of 49 cultural attempts from experimentally infected animals, 42 gave positive cultures *in vitro*. The subcultures of the Temple strain of *Trypanosoma cruzi* have been maintained *in vitro* for over a year and are still infective to susceptible test animals.

4. Sixty-four animals, which were inoculated with the intestinal contents of *Triatoma heidemanni* or trypanosomes derived therefrom, were sacrificed at various intervals. Histopathological studies in these animals revealed 11 cases of intracellular leishmania forms of *Trypanosoma cruzi*. These forms were found in cardiac muscle fibers (7 times), in skeletal muscles (5 times), and in scattered fat cells (once).

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THE ISOLATION AND PATHOGENICITY OF *PITYROSPORUM OVALE*[1]

By C. W. Emmons, *Senior Mycologist, United States Public Health Service*

A small yeast-like microorganism is almost always associated with the dry or greasy scales of seborrhea capitis or dandruff (fig. 2). It can, in fact, be found on the majority of "normal" scalps (*5*) and it is present on other skin surfaces. Rivolta (*14*) is credited by many investigators with being the first to describe this microorganism. It seems doubtful, however, whether the Cryptococcus which he found associated with psoriasis is the same. In the following year Malassez (*6*) described it, and it has been called the spore of Malassez. It is better known as the "bottle bacillus" and *Pityrosporum ovale*.

Although observers agree that *P. ovale* is usually found associated with seborrhea, they do not agree on its cultural characteristics or its etiologic significance. Since the careful but futile efforts of Sabouraud to obtain pure cultures of *P. ovale* many investigators have tried in vain to isolate this fungus. Many of them have observed some growth around the scales used as inoculum, but were unable to obtain subcultures, further growth being inhibited by an inadequate culture medium or by overgrowth of bacteria, yeast, or molds. The claims of some of those who believed they had successfully subcultured this delicate fungus have been later withdrawn or disproved. Attempts to stimulate growth by the addition of substances to the medium usually have not entirely overcome the difficulties encountered. Marzinowski and Bogrow (*7*), Meirowsky (*8*), and Krauss (*4*) added lanolin, and Templeton (*15*) added oleic acid to the media. Panja (*13*) isolated a fungus which he believed was *P. ovale* by placing infected scales on gentian violet glucose agar and then transferring them to 2 percent glycerin agar. Huang (*12*) isolated a strain on agar containing 8 percent glucose and 2 percent lecithin. Ota and Huang (*12*) isolated

[1] From the Division of Infectious Diseases, National Institute of Health.

a second strain on agar containing 10 percent glucose and about the same percentage of butter. Benham (*2*) tested a number of oily materials and found lanolin, oleic acid, and butter most effective in promoting growth.

Ota and Huang and Benham isolated and subcultured fungi which appear to be strains of *P. ovale*. Ota and Huang state that strains isolated by Acton and Panja and by Castellani (*3*) were essentially like their own. The other investigators mentioned, and others, probably also observed growth of the fungus in primary cultures, but were not successful in obtaining subcultures. In view of the recent studies of Benham, and of the studies reported here, the correct identification of some of the more easily cultured strains can well be questioned. The strains of Acton and Panja and of Castellani grew slowly on ordinary media after the first isolation. Some investigators have isolated larger yeast-like forms which are easily subcultured on any of the ordinary mycological media. The strain isolated by Moore (*9*, *10*) and used in inoculation experiments to prove the pathogenicity of *P. ovale* grows readily and quickly on such media. Benham has identified this strain as a member of Group III of Cryptococcus. Species of Cryptococcus are known to be often on the skin.

Aside from the difficulty of obtaining pure cultures for use in experimental inoculations, there is another serious obstacle to obtaining convincing proof that *P. ovale* is pathogenic. It is normally present on nearly all scalps, and adequate fulfillment of Koch's postulates is therefore difficult. Those who claim to have produced seborrhea by experimental inoculation have specified that in order to produce lesions it was necessary in most cases to use individuals who already exhibited lesions of seborrhea, or who had the type of skin usually associated with seborrhea. Since such individuals almost certainly harbored *P. ovale* before the experimental inoculation, its demonstration after inoculation cannot be taken as proof either that the fungus which was used for inoculation produced the seborrhea, or that it was actually a culture of *P. ovale*.

It would appear that in most cases when *P. ovale* has been isolated, assuming that the microorganism obtained was correctly identified, the isolations were largely fortuitous, most attempted cultures yielding no growth or only contaminants. It is, therefore, of interest to report here a method of isolating *P. ovale* which is easy and dependable. Strains of this fungus have been isolated repeatedly by planting untreated scales from the scalp of an individual with seborrhea oleosa (*11*) in dextrose broth (pH 5.5) to which varying amounts of glycerin had been added. The glycerin broths were made up in flasks, tubed, sterilized in the autoclave, and planted by mixing the greasy scales from the scalp as thoroughly as possible with the broth. Growth of *P. ovale* in the lower concentrations of glycerin was inhibited by the

rapid growth of bacteria, but in 28 percent glycerin *P. ovale* grew well and bacterial growth was practically inhibited. Growth of *P. ovale* was not entirely inhibited until the concentration of glycerin reached 48 percent. Table 1 shows the estimated relative amounts of growth of *P. ovale* and of bacteria in various glycerin concentrations after 1 week's incubation at 30° C. Growth was much better at 30°–37° C. than at room temperature.

TABLE 1.—*Estimated relative growth of P. ovale and of bacteria on scales placed in different concentrations of glycerin, after 7 days incubation at 30° C. A small amount of growth was observed in 48 percent glycerin after 2 weeks' incubation*

Percent glycerin	*P. ovale*	Bacteria	Hyphomycete in one or more tubes	Percent glycerin	*P. ovale*	Bacteria	Hyphomycete in one or more tubes
17	++	++++	−	34	+++	−	+
20	+++	+++	+	36	+++	−	+
23	++++	+	+	40	+	−	+
26	++++	±	+	44	±	−	+
28	++++	−	+	48	−	−	−
30	+++	−	+	53	−	−	−
32	+++	−	+				

Growth of *P. ovale* was most easily demonstrated in and around very small scales which floated on the surface of the broth in a dust-like film, but the fungus also grew in the scales which settled to the bottom of the tube. It was at first supposed that the glycerin supplied a nutrient required in the metabolism of the fungus. Further studies indicated, however, that in these isolation cultures the nutrient requirements of the fungus were met by the scales used as inoculum. *P. ovale,* after isolation in pure culture, does not grow readily in any of the concentrations of glycerin found useful in its isolation. The important function of the glycerin is to inhibit the growth of contaminants, particularly of bacteria, which are not as tolerant as the fungus of these high glycerin concentrations. A similar use of glycerin broth may aid in the isolation of other fungi from mycoses. Experiments to test this are planned.

In the experiments made to determine the glycerin tolerance of *P. ovale,* the concentrations to be tested were set up and seeded from portions of the same inoculum in order to minimize differences. Several tubes of each concentration were planted. A few tubes, after incubation, contained Hyphomycetes, growths of these molds appearing in concentrations of glycerin as high as 44 percent. Isolation of *P. ovale* was usually possible even in these contaminated tubes unless the mold produced sprout cells or a fragile, easily torn mycelium.

In view of the conflicting claims for success in subculturing *P. ovale,* proof of the correct identification of any isolate is necessary. Evidence that the yeast-like fungus isolated by this method is actually

P. ovale is supplied by its peculiar nutritional requirements; its morphological resemblance to the budding cells seen in scales from the scalp; and a series of observations, which can readily be made, and which reveal a continuity of development between the budding cells seen in the inoculum and those growing in the cultures (figs. 3–7).

The strains of *P. ovale* isolated by this method, like those isolated by Benham, grow very poorly or not at all on all ordinary media. They grow readily, however, when planted on slants of acid dextrose or wort agar over which an ether extract of either lanolin, oleic acid, or the scales from seborrhea has been pipetted as described by Benham (*2*). My strains have been compared with one which Dr. Benham kindly sent me as typical of hers, and with Dr. Moore's, to

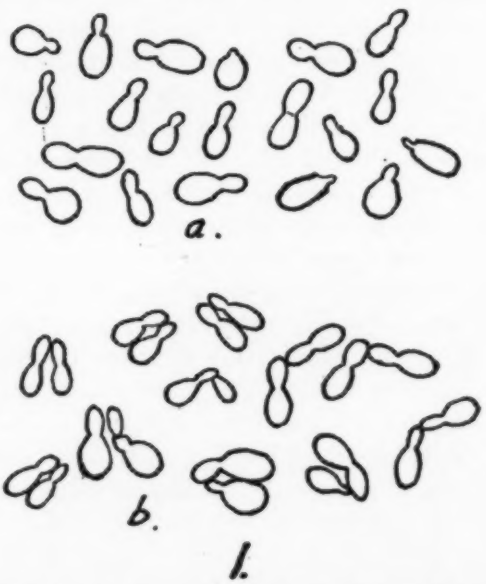

FIGURE 1.—Camera lucida drawings of young cultures of *P. ovale*. 1a, variety of forms found; 1b, tendency to lie side by side or with buds in contact. (×1400)

whom I am also indebted for a culture. The nutritional requirements of my strain are like those of Benham's. My strain grows more slowly and the cells are more uniformly oval and somewhat smaller, but it is probably cospecific with hers. Moore's strain is quite different. It is larger, mucoid, and grows readily on all ordinary media, although cultures are somewhat difficult to maintain unless transferred frequently.

The several strains isolated in glycerin broth have been identical. The cells are small (1.5–2.5 x 2–3.5μ), thin-walled, oval, and bud at one end (figs. 1a, 3, 7). They show a tendency to lie side by side or at an angle with buds in contact (fig. 1b). The position assumed suggests that there may be a conjugation of cells, but this has not

been actually demonstrated. The primary cultures on glycerin broth and, to a slighter extent, subcultures on agar produce a not unpleasant fruity odor suggesting butyl acetate, and similar to that sometimes detectable from the scalp. It is apparently a volatile substance formed through the utilization by *P. ovale* of the fats or fatty acids in the inoculum and on the agar.

Besides the fact that the strains isolated in glycerin broth cannot be subcultured on ordinary media but will grow only when transferred to media covered with a film of some suitable fatty material, and besides the morphological similarity between the fungus isolated in culture and that seen in the scales, a further and perhaps more convincing proof of the identity of the isolate is furnished by following the development of the cells seen in the inoculum. Collected scales are mixed in order to make the inoculum as nearly uniform as possible, and tubes of 23–40 percent glycerin broth are heavily seeded with these scales. An immediate microscopic examination made by mounting some of the scales in broth under a cover slip shows that numerous cells of *P. ovale* are present (fig. 3). The apparent size in glycerin broth is slightly larger (1.5–2.5 x 3.5μ, exclusive of buds) than in xylol, although the cells are not so easily seen. If samples of the inoculum are now examined at intervals of a few hours an increase in numbers of these cells can be clearly demonstrated. The increasing numbers appear at innumerable points *in situ* in the scales, indicating that the microcolonies which develop after a few days around the scales come from the cells of *P. ovale* which were numerous on the scales, and do not arise by the proliferation of one or a few cells of a contaminating yeast which might have been present. Nearly all the small scales which float on the surface of the broth thus become nuclei of microcolonies of actively budding cells.

When the inoculum is examined after 24 to 36 hours incubation many of the individual scales are surrounded, when crushed under a cover slip for microscopic examination, by an oily substance in which an increased number of the cells of *P. ovale* can be seen (fig. 6). Some of the smaller scales have closely associated microcolonies of the organism (fig. 4). After 4 or 5 days incubation the microcolony increases greatly in size, but the scale about which the growth centers can still be seen (fig. 5). In older primary cultures further proliferation of the cells is apparent (fig. 7). These budding cells almost exactly resemble those seen on the original scales, they are only slightly larger, and when transferred to agar media which has become somewhat dry before use, there appears to be no difference in size or appearance. Subcultures are best made by grinding some of the primary culture with broth in a mortar and pipetting onto the previously prepared agar slants.

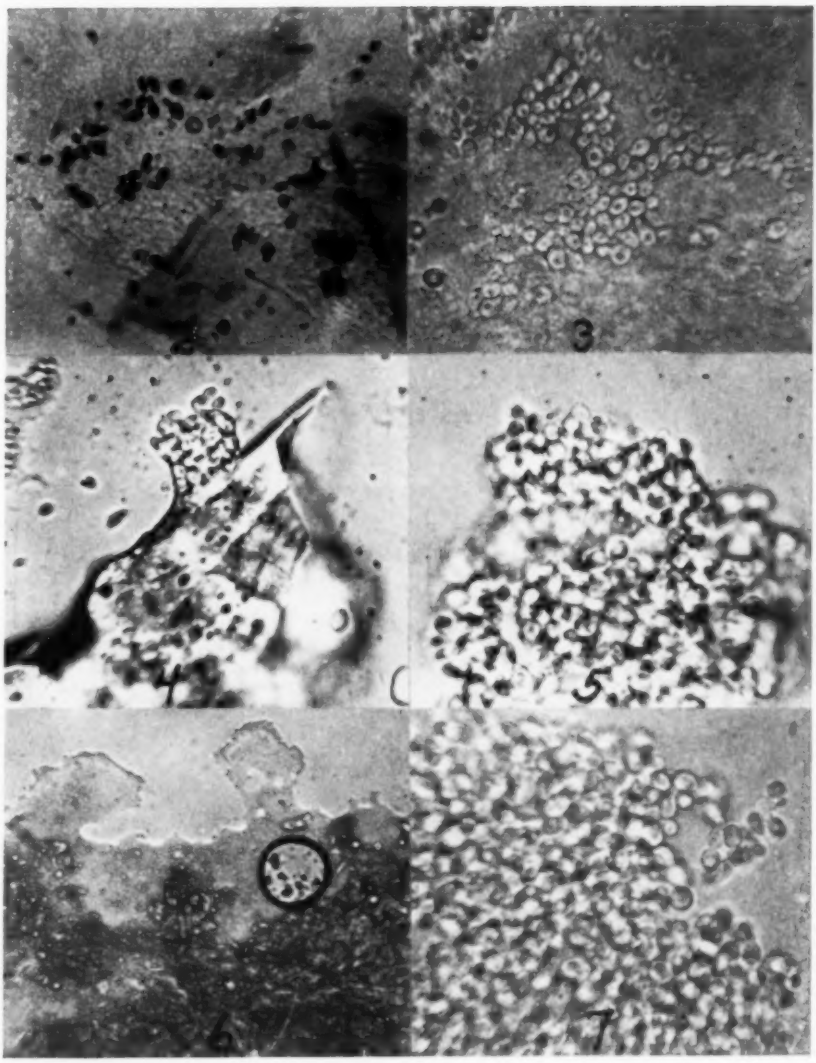

FIGURE 2.—*P. ovale* in defatted, heat-fixed, and methylene blue stained scales from seborrhea. (× 900.)

FIGURE 3.—*P. ovale* in a scale a few hours after planting and before there has been any increase in numbers. Note the apparent increase in size in 28 percent glycerin broth. (× 900.)

FIGURE 4.—A microcolony of *P. ovale* after 54 hours' incubation, showing a close association with the epithelial scale. (× 900.)

FIGURE 5.—A microcolony of *P. ovale* surrounding an epithelial scale after 48 hours' incubation. The size of the colony which can be demonstrated at this stage depends upon the number of *P. ovale* cells in the scale when planted, and the number detached when mounting, as well as upon the time of incubation. (× 900.)

FIGURE 6.—Oily material exuding from epithelial scales incubated 48 hours. The scales were placed in a drop of the broth on a slide and flattened under a cover slip. The oily "fringe" contains a few droplets and numerous cells of *P. ovale*, displaced when the scale was crushed. (× 100.)

FIGURE 7.—Primary culture of *P. ovale* after 5 days incubation. (× 900.)

Attempts were made to determine whether *P. ovale* is pathogenic. The fungus ordinarily does not penetrate to the deeper layers of the skin, being found principally in the horny layers and the superficial layers which line the hair follicle. It should, therefore, be possible to simulate natural infection most closely by thoroughly rubbing a culture into the skin. It is desirable to avoid the trauma incident to intracutaneous injection or scarification because these operations usually result in some scaling and increase in pigmentation. It was further recognized that *P. ovale* is almost universally present on the skin areas subject to seborrhea. Therefore, instead of trying to find and experimentally infect an individual who did not already carry the fungus it was decided to inoculate a seborrheic individual and to measure any noticeable increase in the time required to develop seborrhea in the area inoculated as compared with an uninoculated area. The entire scalp was thoroughly cleaned and a culture from an agar slant covered with a film of lanolin was removed and rubbed vigorously into areas on the scalp and over the shoulders. Lesions of seborrhea did not appear in the inoculated areas over the shoulders, and did not appear any sooner in the inoculated areas on the scalp than in the control areas. These experiments were repeated, but no pathogenic properties of the fungus could be demonstrated. Although some features of seborrhea seem consistent with a parasitic etiology, the failure of these experimental inoculations would give support to the contentions of many dermatologists and medical mycologists, that *P. ovale* is a saprophyte, especially adapted to growth on the skin, but without etiologic significance in seborrhea.

SUMMARY

P. ovale was repeatedly and easily isolated by planting scales from seborrhea oleosa in acid dextrose broth containing 23 to 44 percent glycerin and incubating at 30°–37° C. Subcultures were successfully carried on media prepared by pipetting ether extract of lanolin, oleic acid, or seborrheic scales over agar slants, as described by Benham.

Evidence that the organism was actually *P. ovale* was furnished by the necessity for using special media, the resemblance of the fungus in culture and in the skin, and a series of observations of the inoculum which showed a continuity of development of the cells of *P. ovale* in the scales.

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CHIGGER MITES*

Chigger mites or "chiggers"[1] are the larval forms of various species of mites belonging to the family Trombidiidae, commonly known as harvest mites. Many different species of chiggers are known to attack vertebrate hosts, but only two chigger mites attacking man have been recognized from the United States, one, the common North American chigger,[2] and the other a closely related form found in the northern part of the Mississippi Valley.

Description and distribution.—The chigger or larva of the common North American species is oval, bright red, and, as in the first or larval stage of all mites, possesses only 3 pairs of legs. In the unfed

*A leaflet on this subject is available and may be obtained by addressing the Surgeon General, U. S. Public Health Service, Washington, D. C.

[1] The term "chigger," with variations in spelling (chigoe, jigger, etc.), is also applied to a tropical flea, *Tunga penetrans*, but generally in this country the term is used to designate the larval forms of the trombidiid mites.

[2] Our common North American chigger attacking man is now known under the scientific name of *Leptus rileyi* Oudemans, 1939. In order to aid the reader in tracing the species under its scientific name in both medical and zoological literature, a list of synonyms follows:

Leptus irritans Riley, 1873 (not *Leptus irritans* Lucas, 1847).
Tetranychus tlalsahuate Murray, 1877 (in part).
Trombidium irritans (Riley) Brumpt, 1910.
Trombicula cinnabaris Ewing, 1920.
Leptus similis Hirst, 1921.
Trombicula irritans (Riley) Ewing, 1925.
Trombicula alfreddugèsi (Oudemans, 1910) of Ewing, 1938.
Eutrombicula alfreddugèsi (Oudemans, 1910) of Ewing, 1938.

condition it measures about 150 microns in width, and is scarcely visible to the naked eye. The legs and surface of the body are covered by numerous feathered hairs. The mouthparts consist of a pair of hooked and ventrally barbed fingerlike mandibles, and 2 five-jointed palpi, each of which is provided with a claw divided into 2 prongs at the tip. The adult is a large red hairy mite, with the usual 4 pairs of legs, and with a marked constriction in the anterior portion of the body. Unlike the larval form it is not parasitic but is a scavenger, living largely on the fecal matter of arthropods and on woody decaying substances. Eggs are laid in the ground and the chiggers hatch in the spring soon after warm weather begins.

Chiggers have a widespread distribution in the United States, occurring from Long Island to Mexico and from the Atlantic coast to the Rocky Mountains. They have been found in low lands and well up in the mountains wherever there is rough growth of weeds and shrubbery. They may be encountered from the latter part of April until the last of October, depending upon conditions of temperature and moisture. In the southern United States they may begin to cause annoyance early in May, while in the northern part of their range they seldom appear before the middle of June.

The North American chigger is not only a pest of man but it has been reported as attacking a wide range of vertebrates, including domestic animals, small mammals, birds, and reptiles. It is an important pest of poultry, frequently causing the death of young chickens.

Method of attack.—Chiggers attach themselves to the surface of the skin by means of their mouthparts and feed much as do ticks. They apparently feed upon epidermal tissue liquefied by a secretion which they themselves inject into the skin. When they become fully engorged they drop off. The localization of chigger attachment, to quote one author, is determined by two factors, the tightness of the clothing at certain parts of the body and the thickness of the skin. Experiments by the same writer have shown that chiggers attack by preference where the skin is very thin and the flesh wrinkled or tender. Because of their size, 150 microns in width before they have become engorged, chiggers are unable to enter the pores of the skin (which range from 20 to 50 microns in diameter), but they frequently attach at the mouth of hair follicles. Although it is widely believed that chiggers burrow into the skin and embed their entire body, this method of attack must be extremely uncommon; they would be unable to accomplish such an invasion except in instances where a large enough opening in the skin was already present.

Symptoms.—An intense itching, apparently due to the liquefying secretion injected by the chigger, develops within the first 24 hours after exposure, and this is followed by a breaking out of wheals or

papules surrounded by an inflamed area. The papules may be surmounted by a pinhead-sized vesicle containing clear fluid. The itching generally reaches its maximum on the second or third day, then gradually subsides, though it may persist intermittently for several weeks. Scratching may be followed by secondary infection. If the lesions are numerous, fever, headache, and temporary nervous upset may result, and the intense pruritus may lead to loss of sleep and digestive disturbances. In this country chiggers are not known to transmit any disease, but in the Orient an allied species has been shown to be the carrier of pseudotyphus or Japanese river fever.

Treatment and prevention.—If it is known that there has been exposure to chiggers the skin should be examined, preferably with a hand lens, for the active larvae. However, they are so minute and they move so rapidly over the surface of the skin before attachment that it is difficult to capture them. An application of kerosene or 95 percent alcohol will kill the larvae quite rapidly. As soon as possible after exposure, it is advantageous to apply a thick lather of soap to the affected parts, allowing it to remain for 10 minutes or more before bathing. Even though the larvae may be removed or killed soon after attachment, usually enough secretion has been introduced into the skin to cause the characteristic itching lesion, and for this there is no known specific remedy. The intense itching may be temporarily relieved by ammonia or strong salt water, or a calomel phenol lotion. Collodion with metaphen applied to the lesions is recommended both to relieve the itching and to prevent infection.

In the summer and early fall when it is necessary to go into fields of tall weeds or grass, into berry patches, or wherever there is heavy undergrowth, an efficacious measure to prevent attack by chiggers is the liberal sprinkling of the stockings and underclothing with flowers of sulfur. Some authors have stated that the spraying of the shoes, stockings, and trouser legs with one of the proprietary fly-repellant preparations is successful in warding off attacks by chiggers.

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PLATE I

The expulsion of droplets in an unstifled sneeze. (Photograph reproduced by permission of the Department of Biology and Public Health, Massachusetts Institute of Technology.)

PHOTOGRAPH OF A SNEEZE

Sanitarians have long known that certain diseases are spread by the discharges from the mouth and nose, and that droplet infection plays a role in the dissemination of pathogenic microorganisms. They have also known that such microorganisms may be discharged into the air in greater numbers and to greater distances by the uncovered cough and sneeze than in ordinary breathing. But since such droplets are not visible under ordinary conditions, the risks of infection by this means have not been fully appreciated by the public, and the precautionary warnings of health officers to "cover your cough and your sneeze" are not generally heeded.

If any one has failed to appraise fully the potential danger of spreading infection to others by an uncovered sneeze, he has only to study the accompanying photograph, taken by Prof. M. W. Jennison, of the Department of Biology and Public Health, Massachusetts Institute of Technology, which shows the expulsion of droplets in a violent, unstifled act of sneezing.

According to Dr. C. E. Turner, who furnished the photograph, the picture was taken by the technique of ultra high-speed photography, which substitutes an instantaneous flash of light for the opening and closing of the camera shutter. This stroboscopic light illuminates the object to be photographed with an intense flash of short duration, the light being placed in such a position in this picture as to illuminate the droplets with a dark-field effect, so that they stand out sharply even in daylight and give photographic images larger than actual droplet size. The time of exposure was about 1/30,000 of a second.

In such a sneeze as that illustrated here, the droplets are numbered in the thousands, varying with the intensity of the expiratory effort. The number of bacteria dispersed in a sneeze may also be very large. It is stated that most of the droplets are under 2 mm. in diameter and that many are less than 0.1 mm.

The "muzzle velocity" of some droplets is said to be as great as 150 feet a second, and large droplets may be expelled to a distance of 12 feet, although the majority do not travel more than 2 or 3 feet. The involuntary closing of the mouth near the end of a sneeze tends to form a restricted orifice, resulting in the production of more and smaller droplets, which probably come largely from the saliva in the front of the mouth. Also it is apparent from the photograph that the number of droplets issuing from the nose in an unstifled sneeze is insignificant as compared with the number expelled from the mouth. As stated by Jennison and Edgerton,[1] these observations are

The expulsion of droplets in an unstifled sneeze. (Photograph reproduced by permission of the Department of Biology and Public Health, Massachusetts Institute of Technology.)

[1] Droplet infection of air: High-speed photography of droplet production by sneezing. By M. W. Jennison and H. E. Edgerton, Massachusetts Institute of Technology. Proc. Soc. Exp. Biol. and Med., 43: 455–458 (March 1940).

probably important in relation to infectivity, because of the differences in the microbic flora of the two regions.

Some droplets fall to the floor or ground, while others evaporate, leaving their bacteria suspended in the air, through which they may be disseminated by air currents.

The bacteriologic and epidemiologic aspects of infection of the air were discussed in a recent article by Wells, Wells, and Mudd,[2] who conducted experiments on the concentration of microorganisms in the air. They state that "the numbers of streptococci characteristic of the nasopharynx indicate a hazard of respiratory infection and have a sanitary significance comparable with the presence of *Escherichia coli* in drinking water." They estimate that several thousand nasopharyngeal streptococci per sneeze are contributed to the atmosphere and that "the sneeze thus almost seems to be a provision of nature for the survival of nasopharyngeal parasites. Even where the manifestations of a disease do not provide for the wide autodissemination of the infection through the air it has been observed that an outbreak of colds will be followed by the rapid spread of contagion. Sneezing induced by pollens might conceivably facilitate the spread of nasopharyngeal infection * * *."

Although much is yet to be learned experimentally regarding the physical and other characteristics of expiratory droplets which are factors in determining more accurately the role of droplet transmission in those communicable diseases that are spread by nose and mouth discharges, there can be no question that covering the mouth in coughing and sneezing is an important preventive measure with respect to such diseases.

COURT DECISION ON PUBLIC HEALTH

City ordinance regulating closing hour of barber shops held invalid.—(South Dakota Supreme Court; *City of Huron* v. *Munson*, 289 N. W. 416; decided December 26, 1939.) A complaint, which charged a violation of an ordinance of the city of Huron regulating the hour of closing of barber shops within the city, was dismissed by the trial court, and the city appealed. The power to regulate the business of barbering was not expressly granted to the municipalities of the State, but there was a grant of power to protect the public health. The supreme court said that, if it were conceded that certain general grants of power permitted the regulation of barbering by a city as a means of safeguarding the public health, it did not necessarily follow that the city could regulate the hours during which that business could be carried on. It was pointed out that any such regulation, to come

[2] Infection of air. Bacteriologic and epidemiologic factors. By W. F. Wells, M. W. Wells, and Stuart Mudd. Am. J. Pub. Health, 29: 863-880 (August 1939).

within the scope of the grant of power to protect the public health, had to be reasonable and, to qualify as reasonable, had to contribute in some real and substantial measure to the object sought to be accomplished by the grant of power. Continuing, the court said: "The conceded grant of power has as its purpose the protection of public health. We are convinced that the hour of closing a barber shop bears no real or substantial relation to that purpose, and that such a regulation contained in a city ordinance is therefore invalid as beyond the scope of the power granted by the legislature."

DEATHS DURING WEEK ENDED JULY 6, 1940

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended July 6, 1940	Corresponding week, 1939
Data from 88 large cities of the United States:		
Total deaths	7,116	7,142
Average for 3 prior years	7,394	
Total deaths, first 27 weeks of year	238,492	236,561
Deaths under 1 year of age	426	444
Average for 3 prior years	513	
Deaths under 1 year of age, first 27 weeks of year	13,669	14,022
Data from industrial insurance companies:		
Policies in force	65,119,180	67,112,141
Number of death claims	8,858	8,512
Death claims per 1,000 policies in force, annual rate	7.1	6.6
Death claims per 1,000 policies, first 27 weeks of year, annual rate	10.1	10.9

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED JULY 13, 1940

Summary

The incidence of poliomyelitis continues to attract interest despite the favorable trend that has been evidenced throughout the current season. A report of 101 cases for the week ended July 13 compares favorably with 143 cases for the corresponding week in 1939 which was also the median week for the 1935–39 period. For the current week California reported 27 cases and Washington 17. The other 57 cases were scattered among 23 States.

Typhoid fever increased from 215 cases for the preceding week to 238 cases, the largest numbers being reported from Arkansas, Louisiana, and Texas. The typhoid trend for 1940 has been below the seasonal expectancy and lower than the 1939 incidence for each week of the year.

Slight increases were noted in the incidence of diphtheria, influenza, measles, meningitis, scarlet fever, smallpox, and whooping cough; however, with the exception of measles, the incidence of all the common communicable diseases was below the 1935–39 median figure for the corresponding week.

Eighteen cases of Rocky Mountain spotted fever were reported, of which 17 were in the Central and Eastern States. The 34 cases of typhus fever reported were scattered among 8 South Atlantic and South Central States.

Telegraphic morbidity reports from State health officers for the week ended July 13, 1940, and comparison with corresponding week of 1939 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39
	July 13, 1940	July 15, 1939		July 13, 1940	July 15, 1939		July 13, 1940	July 15, 1939		July 13, 1940	July 15, 1939	
NEW ENG.												
Maine	1	1	1	------	------	------	141	50	50	0	0	0
New Hampshire	0	1	0	------	------	------	0	7	3	0	0	0
Vermont	0	1	0	------	------	------	8	76	37	0	0	0
Massachusetts	2	3	4	------	------	------	774	410	217	0	1	1
Rhode Island	0	1	1	------	------	------	59	52	17	0	0	0
Connecticut	0	0	3	1	2	1	8	108	53	0	0	0
MID. ATL.												
New York	15	21	26	[1] 2	[1] 6	[1] 3	681	840	1,066	1	0	10
New Jersey [2]	9	7	7	4	------	2	749	20	247	0	0	1
Pennsylvania	9	22	17	------	------	------	245	66	480	1	4	3
E. NO. CEN.												
Ohio	8	6	13	6	13	7	12	77	233	0	2	3
Indiana	2	10	9	------	12	8	9	6	10	1	4	1
Illinois [2]	25	17	22	2	10	10	256	23	91	0	0	4
Michigan [2]	1	6	14	6	------	------	370	98	137	1	0	1
Wisconsin	0	1	3	9	14	14	621	190	190	2	0	0
W. NO. CEN.												
Minnesota	1	1	2	------	1	1	18	29	53	0	0	0
Iowa	0	4	4	3	1	------	35	55	15	1	0	0
Missouri	0	3	9	------	------	27	2	3	16	0	0	1
North Dakota	0	2	1	------	64	9	0	39	8	0	0	1
South Dakota	0	1	2	------	------	------	0	15	3	0	0	0
Nebraska	1	4	2	------	------	------	13	8	8	1	0	0
Kansas	4	0	3	1	------	2	53	10	10	1	0	1
SO. ATL.												
Delaware	0	0	1	------	------	------	0	1	2	0	0	0
Maryland [2] [3]	0	2	3	1	5	2	4	27	27	0	0	3
Dist. of Col [2]	5	5	6	------	1	------	1	35	33	0	0	0
Virginia [2]	4	11	7	36	19	------	36	91	60	1	3	4
West Virginia [2]	2	3	3	2	11	7	6	2	28	2	0	1
North Carolina [2] [4]	2	7	10	------	1	1	48	82	82	1	2	3
South Carolina [4]	3	6	3	105	84	40	6	8	8	0	2	1
Georgia [4]	2	10	9	28	26	------	15	15	0	0	0	1
Florida [4]	3	2	3	2	7	------	16	11	8	0	1	1
E. SO. CEN.												
Kentucky	1	4	4	4	------	2	42	2	15	1	3	2
Tennessee	2	3	3	12	13	9	25	22	19	1	0	2
Alabama [4]	1	10	10	7	9	9	53	8	10	1	0	0
Mississippi [3] [4]	8	3	3	------	------	------	------	------	------	0	0	1
W. SO. CEN.												
Arkansas	2	5	5	1	6	4	16	23	6	1	2	2
Louisiana [4]	4	4	7	10	31	18	1	------	9	1	1	1
Oklahoma [2]	4	1	3	13	3	7	4	20	14	1	0	0
Texas [4]	13	26	20	44	87	67	125	85	76	1	0	2
MOUNTAIN												
Montana [2]	0	0	1	------	------	------	22	29	29	0	1	0
Idaho	1	0	0	------	------	1	12	2	5	0	0	0
Wyoming	0	0	0	------	------	------	12	21	2	0	0	0
Colorado [3]	5	5	3	------	------	------	10	16	20	0	0	0
New Mexico	0	0	0	------	------	------	10	4	5	0	0	0
Arizona	0	0	1	24	10	9	41	4	5	0	0	0
Utah [3]	0	1	1	------	------	------	69	24	24	0	0	0
PACIFIC												
Washington	1	0	0	------	------	------	48	368	92	0	0	0
Oregon	1	1	1	1	6	4	35	61	18	0	0	0
California	10	21	20	5	17	17	129	479	397	2	1	3
Total	152	242	307	329	459	374	4,840	3,622	3,912	22	27	79
28 weeks	8,050	10,666	12,796	167,313	150,230	140,743	217,367	342,249	342,249	992	1,232	3,795

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended July 13, 1940, and comparison with corresponding week of 1939 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39	Week ended		Median, 1935–39
	July 13, 1940	July 15, 1939		July 13, 1940	July 15, 1939		July 13, 1940	July 15, 1939		July 13, 1940	July 15, 1939	
NEW ENG.												
Maine	0	0	0	3	56	10	0	0	0	2	0	1
New Hampshire	0	0	0	1	0	3	0	0	0	0	1	0
Vermont	0	0	1	2	1	2	0	0	0	0	6	1
Massachusetts	0	1	1	66	51	66	0	0	0	2	4	3
Rhode Island	0	0	0	4	0	6	0	0	0	2	0	0
Connecticut	2	0	0	26	13	13	0	0	0	5	3	3
MID. ATL.												
New York	1	6	6	181	103	155	0	0	0	1	13	14
New Jersey [3]	0	2	2	110	31	31	0	0	0	8	6	6
Pennsylvania	0	0	0	120	98	144	0	0	0	14	6	14
E. NO. CEN.												
Ohio	1	5	1	52	91	91	0	12	0	6	9	12
Indiana	3	1	1	7	18	23	1	2	2	0	8	8
Illinois [3]	0	5	5	206	69	87	1	3	11	9	25	23
Michigan [3]	4	5	2	102	85	129	0	1	1	6	1	3
Wisconsin	1	2	0	53	42	66	2	0	5	0	0	1
W. NO. CEN.												
Minnesota	0	6	1	24	13	34	0	2	6	3	0	0
Iowa	5	0	0	10	13	19	11	13	6	1	2	2
Missouri	0	1	1	5	8	19	1	3	5	4	5	11
North Dakota	0	2	0	3	2	10	5	2	2	1	0	0
South Dakota	0	0	0	5	4	4	16	7	5	0	0	0
Nebraska	0	1	0	3	5	5	1	3	3	1	0	1
Kansas	4	0	0	25	23	27	0	1	3	1	0	5
SO. ATL.												
Delaware	1	0	0	3	2	2	0	0	0	0	1	1
Maryland [3] [5]	0	0	0	7	16	16	0	0	0	4	2	12
Dist. of Col [3]	0	0	0	8	1	3	0	0	0	0	4	3
Virginia [3]	1	1	3	10	14	8	0	0	0	8	37	18
West Virginia [3]	2	0	0	16	9	12	0	0	0	5	17	9
North Carolina [4] [5]	2	3	3	16	9	15	1	2	0	4	19	21
South Carolina [4]	3	20	1	0	1	2	0	0	0	10	21	21
Georgia [4]	0	5	1	4	1	5	0	0	0	15	24	39
Florida [4]	0	3	0	2	5	2	0	0	0	4	1	1
E. SO. CEN.												
Kentucky	3	3	1	14	4	10	0	0	0	8	37	37
Tennessee	0	2	7	5	15	4	0	0	0	3	32	42
Alabama [4]	5	2	3	10	10	10	1	0	0	2	13	20
Mississippi [3] [4]	0	0	1	1	2	3	0	0	0	5	9	11
W. SO. CEN.												
Arkansas	2	1	0	5	1	6	0	1	0	25	13	23
Louisiana [4]	3	1	1	6	7	6	0	0	0	22	40	21
Oklahoma [3]	2	1	1	3	5	7	2	0	1	8	20	20
Texas [4]	7	15	1	8	17	17	3	0	0	33	30	30
MOUNTAIN												
Montana [3]	1	0	0	3	8	8	1	0	1	1	1	1
Idaho	0	0	0	0	2	3	0	0	2	0	2	2
Wyoming	0	0	0	3	7	5	0	2	2	0	0	0
Colorado [5]	0	1	0	16	9	21	1	2	3	2	5	2
New Mexico	1	1	0	1	4	5	0	0	0	1	6	5
Arizona	0	1	0	1	1	2	0	2	0	5	2	4
Utah [3]	1	1	0	4	4	9	0	0	0	0	1	1
PACIFIC												
Washington	17	0	0	14	5	14	1	1	4	3	2	3
Oregon	2	0	0	4	5	10	1	1	3	1	2	3
California	27	45	19	53	66	80	0	14	7	3	7	11
Total	101	143	143	1,225	956	1,391	49	74	103	238	437	520
28 weeks	945	1,020	1,020	115,292	112,675	160,214	1,843	8,454	7,557	3,099	4,601	4,839

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended July 13, 1940, and comparison with corresponding week of 1939 and 5-year median—Con.

Division and State	Whooping cough	
	Week ended	
	July 13, 1940	July 15, 1939
NEW ENG.		
Maine	12	26
New Hampshire	0	0
Vermont	16	47
Massachusetts	105	140
Rhode Island	2	31
Connecticut	63	53
MID. ATL.		
New York	265	413
New Jersey [2]	142	239
Pennsylvania	357	438
E. NO. CEN.		
Ohio	270	524
Indiana	12	98
Illinois [2]	157	362
Michigan [3]	261	181
Wisconsin	108	212
W. NO. CEN.		
Minnesota	43	35
Iowa	7	34
Missouri	33	36
North Dakota	9	58
South Dakota	6	3
Nebraska	6	34
Kansas	61	22
SO. ATL.		
Delaware	11	7
Maryland [2 3]	144	65
Dist. of Col [3]	13	38
Virginia [2]	110	58
West Virginia [3]	91	8
North Carolina [2 4]	121	274
South Carolina [4]	15	18
Georgia [4]	20	34
Florida [4]	10	33
E. SO. CEN.		
Kentucky	92	44
Tennessee	48	130
Alabama [4]	17	21
Mississippi [3 4]	----	----
W. SO. CEN.		
Arkansas	36	15
Louisiana [4]	64	159
Oklahoma [3]	19	4
Texas [4]	210	115
MOUNTAIN		
Montana [2]	8	6
Idaho	14	0
Wyoming	6	1
Colorado [5]	11	38
New Mexico	18	19
Arizona	0	0
Utah [4]	117	76
PACIFIC		
Washington	65	17
Oregon	28	20
California	242	109
Total	3,465	4,295
28 weeks	90,001	109,344

[1] New York City only.
[2] Rocky Mountain spotted fever, week ended July 13, 1940, 17 cases as follows: New Jersey, 2; Illinois, 2; Maryland, 2; District of Columbia, 1; Virginia, 3; North Carolina, 3; Oklahoma, 4; Montana, 1.
[3] Period ended earlier than Saturday.
[4] Typhus fever, week ended July 13, 1940, 34 cases as follows: North Carolina, 3; South Carolina, 5; Georgia, 9; Florida, 2; Alabama, 4; Mississippi, 1; Louisiana, 4; Texas, 6.
[5] Colorado tick fever, week ended July 13, 1940, Colorado, 2 cases.

PLAGUE INFECTION IN LICE FROM A MARMOT IN PARK COUNTY, WYO.

Under date of July 5, 1940, Surgeon L. B. Byington reported plague infection proved in a pool of 14 lice from 1 marmot (*Marmota flaviventris*) shot 12 miles northwest of Cody, Park County, Wyo., on June 17. This is stated to be the first proof of plague infection in that county.

WEEKLY REPORTS FROM CITIES

City reports for week ended June 29, 1940

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	114	31	15	2,433	337	787	9	366	47	1,243	----
Current week [1]	49	32	13	2,519	232	616	1	364	34	935	----
Maine:											
Portland	0	----	0	10	4	0	0	1	0	3	33
New Hampshire:											
Concord	0	----	0	0	0	0	0	0	0	0	7
Manchester	0	----	0	0	0	2	0	0	0	0	9
Nashua	0	----	0	0	0	0	0	0	0	0	4
Vermont:											
Barre	----	----	----	----	----	----	----	----	----	----	----
Burlington	0	----	0	0	0	0	0	0	0	0	12
Rutland	0	----	0	0	0	0	0	0	0	0	2
Massachusetts:											
Boston	0	----	0	165	6	28	0	11	2	60	189
Fall River	0	----	0	89	1	0	0	1	0	9	21
Springfield	0	----	0	3	2	1	0	1	0	0	40
Worcester	0	----	0	234	4	1	0	3	0	2	49
Rhode Island:											
Pawtucket	0	----	0	0	0	0	0	0	0	0	19
Providence	0	----	0	66	0	1	0	0	0	6	50
Connecticut:											
Bridgeport	0	----	0	6	1	1	0	1	0	0	31
Hartford	0	----	0	0	5	7	0	1	0	2	43
New Haven	0	----	0	3	2	6	0	0	0	14	38
New York:											
Buffalo	0	----	0	0	4	15	0	3	0	2	101
New York	16	5	2	368	32	131	0	71	5	102	1,268
Rochester	0	----	0	4	4	2	0	2	0	6	71
Syracuse	0	----	0	0	0	1	0	1	0	4	31
New Jersey:											
Camden	1	----	0	6	5	8	0	0	1	0	23
Newark	0	6	0	248	3	11	0	7	0	6	74
Trenton	0	----	0	0	3	2	0	4	0	0	40
Pennsylvania:											
Philadelphia	2	----	0	173	10	51	0	28	5	36	357
Pittsburgh	3	----	0	1	6	5	0	6	1	33	146
Reading	0	----	0	1	2	0	0	2	0	15	27
Scranton	1	----	----	0	----	1	0	----	0	0	----
Ohio:											
Cincinnati	0	----	0	1	4	4	0	4	0	16	132
Cleveland	1	8	2	4	4	11	0	10	0	48	163
Columbus	0	----	0	0	1	4	0	4	0	8	58
Toledo	1	----	0	1	2	10	0	4	0	18	61
Indiana:											
Anderson	0	----	0	0	2	0	0	0	0	2	6
Fort Wayne	0	----	0	4	1	0	0	1	0	4	17
Indianapolis	0	----	3	2	4	2	0	2	0	4	109
Muncie	0	----	0	0	1	0	0	0	0	0	18
South Bend	0	----	1	0	3	0	0	0	0	0	19
Terre Haute	0	----	0	0	0	0	0	2	0	0	19
Illinois:											
Alton	0	----	0	0	1	0	0	1	0	0	8
Chicago	8	----	0	152	15	171	0	41	1	44	630
Elgin	0	----	0	0	0	0	0	0	0	4	12
Moline	0	----	0	1	0	0	0	0	0	1	8
Springfield	0	----	0	0	3	0	0	1	0	3	19
Michigan:											
Detroit	0	----	0	296	6	29	0	11	1	65	234
Flint	0	----	0	1	0	3	0	2	0	0	20
Grand Rapids	0	----	0	16	1	7	0	2	0	14	31
Wisconsin:											
Kenosha	0	----	0	29	0	2	0	0	0	1	6
Madison	0	----	0	40	0	0	0	1	0	1	11
Milwaukee	0	----	0	335	0	19	0	3	0	9	83
Racine	0	----	0	15	0	0	0	0	0	2	13
Superior	0	----	0	26	0	1	0	0	0	0	6

[1] Figures for Barre and Minneapolis estimated; reports not received.

City reports for week ended June 29, 1940—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth	0		0	9	1	1	0	0	0	1	15
Minneapolis											
St. Paul	0		0	1	2	3	0	1	0	4	57
Iowa:											
Cedar Rapids	0			2		1	0		0	3	
Davenport	0			0		0	0		0	0	
Des Moines	0		0	3	0	4	2	0	0	0	29
Sioux City	0			0		3	0		0	1	
Waterloo	1			2		0	0		0	3	
Missouri:											
Kansas City	0		1	3	4	5	0	3	0	3	88
St. Joseph	0		0	0	0	1	0	0	0	0	14
St. Louis	0		0	4	6	4	0	10	1	10	183
North Dakota:											
Fargo	0		0	0	0	1	0	0	0	0	13
Grand Forks	0			0		1	0		0	5	
Minot	0		0	0	0	0	0	0	0	0	6
South Dakota:											
Aberdeen	0			0		0	0		0	7	
Nebraska:											
Lincoln	0			0		1	0		0	1	
Omaha	1		0	2	0	0	0	3	1	1	50
Kansas:											
Lawrence	0	1	0	0	0	0	0	0	0	3	3
Topeka	0		0	11	2	2	0	0	0	0	21
Wichita	0	1	0	0	1	0	0	1	1	2	30
Delaware:											
Wilmington	0		0	1	0	2	0	0	1	3	20
Maryland:											
Baltimore	0	1	1	4	8	3	0	10	0	122	192
Cumberland	0		0	0	0	0	0	0	0	0	11
Frederick	0		0	0	0	0	0	0	0	0	6
Dist. of Col.:											
Washington	0		0	1	3	11	0	12	0	1	152
Virginia:											
Lynchburg	1		0	2	1	0	0	1	0	5	17
Norfolk	0		0	9	1	0	0	1	0	0	15
Richmond	0		0	0	1	0	0	0	0	1	38
Roanoke	0		0	17	0	0	0	0	0	3	10
West Virginia:											
Charleston	0		0	0	2	3	0	1	0	1	15
Huntington	0			0		0	0		0	0	
Wheeling	0		0	0	2	0	0	0	0	1	24
North Carolina:											
Gastonia	0			0	0		0	0		1	
Raleigh	0		0	0	1	0	0	0	0	3	22
Wilmington	0		0	0	2	0	0	0	0	0	12
Winston-Salem	0		0	0	0	1	0	2	0	4	21
South Carolina:											
Charleston	0		0	0	1	1	0	1	0	0	21
Florence	0		0	0	0	0	0	0	0	0	19
Greenville	1		0	0	0	0	0	0	1	1	4
Georgia:											
Atlanta	0	3	0	1	7	1	0	5	0	18	72
Brunswick	0		0	0	0	0	0	0	0	0	2
Savannah	0		0	0	0	0	0	3	1	1	25
Florida:											
Miami	0		0	1	3	1	0	4	1	0	40
Tampa	0		0	9	1	0	0	1	0	0	29
Kentucky:											
Ashland	0		0	0	1	0	0	0	0	1	10
Covington	0		0	5	1	0	0	4	0	5	18
Lexington	0		0	40	0	0	0	1	0	3	15
Louisville	0		0	6	2	5	0	1	0	32	62
Tennessee:											
Knoxville	0		0	5	0	1	0	0	0	0	27
Memphis	0		0	4	2	0	1	4	1	9	80
Nashville	0		0	3	3	2	0	3	1	8	57
Alabama:											
Birmingham	0		1	5	4	2	0	4	1	2	58
Mobile	0		0	0	0	2	0	1	0	0	18
Montgomery	1			0		0	0		0	0	
Arkansas:											
Fort Smith	0			0		0	0		0	2	
Little Rock	0		0	0	2	0	0	1	1	0	5

City reports for week ended June 29, 1940—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Louisiana:											
New Orleans	0	------	0	3	6	1	0	12	3	32	140
Shreveport	0	------	0	0	0	0	0	1	3	0	42
Oklahoma:											
Oklahoma City	0	------	0	1	1	1	0	3	0	0	33
Tulsa	0	------	0	2	5	0	0	1	1	9	20
Texas:											
Dallas	2	------	0	34	2	0	0	3	2	7	78
Fort Worth	0	------	0	0	2	1	0	1	0	0	37
Galveston	0	------	0	0	0	0	0	2	0	0	25
Houston	0	1	0	14	4	2	0	7	1	1	110
San Antonio	0	------	0	0	3	0	0	5	0	8	63
Montana:											
Billings	0	------	0	0	0	0	0	1	0	0	7
Great Falls	0	------	0	18	3	0	0	0	0	0	7
Helena	0	------	0	0	0	0	0	0	0	0	4
Missoula	0	------	0	1	1	0	0	0	0	0	9
Idaho:											
Boise	0	------	0	0	0	0	0	1	0	0	5
Colorado:											
Colorado Springs	0	------	0	1	1	2	0	1	0	0	0
Denver	8	------	0	12	5	2	0	4	0	0	92
Pueblo	0	------	0	4	1	0	0	0	0	3	8
New Mexico:											
Albuquerque	0	------	0	0	0	0	0	2	0	2	12
Utah:											
Salt Lake City	0	------	0	52	2	3	0	0	0	54	38
Washington:											
Seattle	0	------	0	33	3	3	0	3	0	8	78
Spokane	0	------	0	1	0	0	0	0	0	0	25
Tacoma	0	------	0	1	0	1	0	1	0	0	33
Oregon:											
Portland	2	------	0	7	3	3	0	0	0	10	76
Salem	0	------	------	0	------	0	0	------	0	0	------
California:											
Los Angeles	3	6	0	6	1	12	0	23	0	77	326
Sacramento	0	------	0	1	1	4	0	1	0	1	30
San Francisco	3	1	2	3	3	4	0	5	0	16	161

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
Rhode Island:				District of Columbia:			
Providence	1	0	0	Washington	1	1	1
New York:				Virginia:			
Buffalo	3	1	0	Richmond	0	0	1
New York	2	0	0	Oklahoma:			
Illinois:				Oklahoma City	0	0	1
Chicago	0	0	1	Washington:			
Wisconsin:				Tacoma	0	0	10
Milwaukee	1	0	0	California:			
Missouri:				Los Angeles	0	0	5
St. Joseph	0	1	0	San Francisco	0	0	1
Nebraska:							
Omaha	0	0	1				

Encephalitis, epidemic or lethargic.—Cases: New York, 1; St. Louis, 1; Omaha, 1; Missoula, 1.
Pellagra.—Cases: Birmingham, 1.
Typhus fever.—Cases: Charleston, S. C., 1; Miami, 1; New Orleans, 2. Deaths: New York, 1.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended June 8, 1940.—During the week ended June 8, 1940, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis				1	5					6
Chickenpox		20	1	197	439	35	9	2	79	782
Diphtheria				34	1	1				36
Dysentery				1						1
Influenza		8			51				18	77
Measles			5	116	289	158	146	2	94	810
Mumps				45	272	7	40		29	393
Pneumonia		3			17	2			5	27
Poliomyelitis					1	1				2
Scarlet fever		2	1	80	100	15	13	12		223
Trachoma									2	2
Tuberculosis	3	2	9	38	41	31	15	4		143
Typhoid and paratyphoid fever			1	13	2	1	5	1	1	24
Whooping cough		48	3	125	112	29	36	4	25	382

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE:—A cumulative table giving current information regarding the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS of June 28, 1940, pages 1188-1191. A similar table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Plague

China—South Hsingan Province—Tungliao (vicinity of).—A report dated July 7, 1940, stated that up to July 6, 1940, 17 cases of plague had occurred in the vicinity of Tungliao, South Hsingan Province, China.

Hawaii Territory—Island of Hawaii—Hamakua District—Paauilo (vicinity of).—A rat found on June 7, 1940, another on June 18, and another on June 20, 1940, in the vicinity of Paauilo, Hamakua District, Island of Hawaii, T. H., have been proved positive for plague.

United States—Wyoming—Park County.—A report of plague infection in Park County, Wyoming, appears on page 1319 of this issue of PUBLIC HEALTH REPORTS.

Typhus Fever

Straits Settlements—Singapore.—During the week ended May 4, 1940, 1 case of typhus fever was reported in Singapore, Straits Settlements.

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